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published in
Behavior Genetics
1989

DOI (link to publisher)
10.1007/BF01065888

document version
Publisher's PDF, also known as Version of record

Behavior Genetics, Vol. 19, No. 1, 1989

Resemblances of Parents and Twins in Sports Participation and Heart Rate

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A model to analyze resemblances of twins and parents using LISREL is outlined and applied to sports participation and heart-rate data. Sports participation and heart rate were measured in 44 monozygotic and 46 dizygotic adolescent twin pairs and in their parents. Genetic factors influence variation in both sports behavior and heart rate, while there is no evidence for transmission from parental environment to offspring environment. For sports participation the data support a model in which there is a high positive correlation between environments of spouses and between environments of female twins. This correlation is absent for male twins and negative for opposite sex twins. For heart rate, a positive correlation between environmental influences was observed for all twins; there is no evidence for assortative mating. The proposed model can also handle data sets where parents and twins have been measured on more than one variable. This is illustrated by an application to the observed association of sports participation and heart rate.

KEY WORDS: twins; parents; sports participation; heart rate; LISREL/PRELIS; discrete variables.

INTRODUCTION

Several studies show evidence for familial influences on sports participation (e.g., Lewko and Greendorfer, 1978; Snyder and Spreitzer, 1973,

This work was aided by NATO Grant 86/0823 and grants from the Belgian National Research Fund, the State University of Gent, and the Catholic University of Leuven. We would like to thank Drs. R. Frants and I. Hendriks for zygosity typing.

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0001-82444/89/0100-0123$06.00/0

1976). Lewko and Greendorfer (1978) conclude in a review that the family rather than schools and peers influences sports involvement in children and that within families parents are more influential than siblings in socializing their children into sports participation. Snyder and Spreitzer (1973) note a similarity between spouses in sports behavior. It is unknown, however, whether this resemblance lies in mate selection processes or in transmission of an interest in sports of one partner to the other. It is also unknown whether the observed familial influences are mainly environmental or mainly genetic. The influence of genetic and environmental factors on variation in sports participation has not been studied. Most research, including research with twins, has been directed to sports performance or correlates of performance (e.g., Malina and Bouchard, 1986).

Sports participation is known to influence heart rate: resting heart rate generally is lower in people who frequently engage in sports activities. Twin studies indicate that variation in heart-rate level is explained by genetic as well as shared environmental factors (e.g., Boomsma and Gabrielli, 1985). In this paper an extended twin design is used to partition variation in sports participation and heart rate into genetic and environmental components and to study the association of sports participation and heart rate.

One approach to model data from twins and parents is outlined by Eaves *et al.* (1989) in this issue. They adopt the social homogamy model of Rao *et al.* (1974) in which an observed correlation between spouses is due to their assortment for the cultural environment. Resemblances of parents and children are modeled by a path from parental genotype to offspring genotype and by a path from cultural environment of parents to cultural environment of children. Eaves *et al.* formulate a LISREL model in which data from parents are represented as independent X variables and offspring data as dependent Y variables. We use a different model (shown in Fig. 1) and LISREL formulation. In this model the impact of total parental environment on offspring environment is considered (Vogler and Fulker, 1983) and assortment of spouses is for the total environment. The total offspring environmental correlation, i.e., the whole effect of shared environment in twins (Fulker, 1982), is partly accounted for by parental influences: $2z^2[1 + \psi(s)]$, where $\psi(s)$ is the correlation between the total environments of spouses and z is the environmental transmission parameter. Separate paternal and maternal influences are possible, but as Eaves *et al.* (1989) have shown that these are very difficult to separate, we consider only one environmental transmission parameter for both parents. The other part of the environmental correlation between twins $[\psi(t)]$ is independent of their resemblance with their parents.

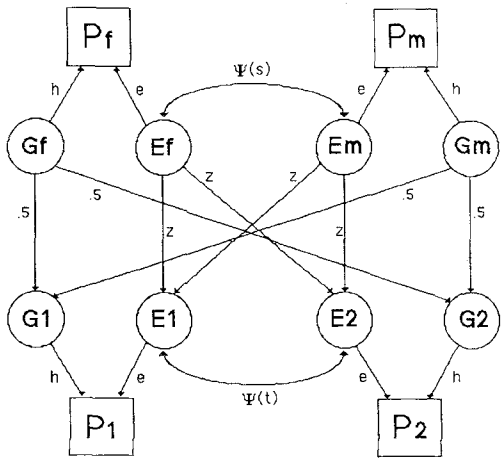

Fig. 1. Parent-offspring model for LISREL analysis. *P* are observed phenotypes of father (F), mother (M), Twin 1, and Twin 2. *G* and *E* denote latent genotype and total environment. Nongenetic transmission (*z*) is from total parental environment to offspring environment and is modeled here to be equal for fathers and mothers. The correlation between total environments of spouses is represented by $\psi(s)$ and the correlation between residual environments of twins by $\psi(t)$.

Based on this model, equations for expected correlations become the following: spouses, $\psi(s)e^2$; parent–offspring, $.5h^2 + (ze^2)[1 + \psi(s)]$; dizygotic (DZ) twins, $.5h^2 + 2(z^2e^2)[1 + \psi(s)] + \psi(t)e^2$; monozygotic (MZ) twins, $h^2 + 2(z^2e^2)[1 + \psi(s)] + \psi(t)e^2$.

This model can be represented in LISREL (Jöreskog and Sörbom, 1986b) using Y and η variables only. This representation makes generalizations to extended data sets possible, such as, for example, to data from more than two generations. A small simulation study using the above model in LISREL is presented below and next the model is applied to measures of sports participation and heart rate.

The sports participation variable measured in this study is dichotomous, the answer to the question "Have you been involved in sports activities during the last three months?" being either "yes" or "no." Such dichotomous traits may be regarded as expressions of an underlying continuous normal distribution (Falconer, 1981). The trait is expressed only if an individual's value on this distribution exceeds some threshold. Maximum-likelihood estimates of sample thresholds and polychoric correlations among values on the underlying distribution can be obtained from LISREL VI (mainframe) or from PRELIS (PC) (Jöreskog and Sörbom, 1986a), as well as polyserial correlations among dichotomous and continuous variables such as sports participation and heart rate.

SUBJECTS

Subjects were 90 twin pairs between 14 and 20 years of age and their parents, who participated in a larger ongoing project on genetic aspects of cardiovascular risk factors. Addresses of twin pairs living in Amsterdam (71) were obtained from the population registry of the City Council. In addition, 19 twin pairs and their parents from outside Amsterdam also participated in the study. Zygosity of the twins was determined by blood typing and, in four cases, also by DNA fingerprinting (Jeffreys *et al.*, 1985). There were 16 monozygotic (MZ) male and 28 MZ female pairs and 15 dizygotic (DZ) male, 17 DZ female, and 14 DZ opposite-sex (OS) twin pairs.

Sports participation and heart-rate data were obtained when subjects visited the laboratory. Heart-rate data were obtained from an electrocardiogram (ECG) that was recorded for an 8.5-min period of rest. Subjects were seated in a comfortable chair in a sound-attenuated cabin and were asked to relax as much as possible. The ECG signal was digitized at 250 Hz and these data were used to determine interbeat intervals (IBIs). These represent the time in milliseconds between successive R waves in the ECG (as IBI increases, heart rate goes down).

ANALYSIS

Data were analyzed by LISREL-PC and PRELIS and by LISREL VI, mainframe. PRELIS is a preprocessor for LISREL-PC and available for personal computers. In LISREL VI it takes two options to specify that one of the variables is discontinuous: MA = KM, the type of matrix to be used for data analysis, is a correlation matrix and MV = 2, the maximum number of distinct values in the discrete variable is two—variables with higher values are analyzed as being continuous. In PRELIS these specifications are MC = 2 and MA = PM to obtain a matrix of polychoric, polyserial, and product–moment correlations. When MA = KM is used in PRELIS, ordinal variables are transformed to normal scores before product–moment correlations among all variables are computed. When MA = KM is used without declaring ordinal variables as ordinal, product–moment correlations on the raw scores are computed.

PRELIS requires a group size of at least 20 to compute polychoric and polyserial correlations. This requirement can be relaxed by specifying on the OU line MS = N (where N is minimum sample size), but for smaller groups the correlations may become unreliable. Therefore, we also computed phi coefficients among the dichotomous variables and compared these to the tetrachoric correlations (Lord and Novick, 1968, p. 347). Phi is a measure of association for "true" dichotomous variables that are not

based on an underlying continuum. Since it is equivalent to the product–moment correlation applied to a 2 × 2 table, phi is easily computed by PRELIS by just leaving out the MC = 2 specification.

Under assumptions of bivariate normality, tetrachoric correlations are independent of estimated thresholds, whereas phi depends on the point of dichotomization. For this reason phi coefficients are not recommended for use in factor analysis (Lord and Novick, 1968) and model fitting in this paper is carried out on tetrachoric correlation matrices.

There is no guarantee that a matrix of sample polychoric and polyserial correlations will be nonsingular, even when the population matrix is Gramian. When a nonpositive definite matrix is obtained, maximum-likelihood (ML) estimation cannot be used and unweighted least squares (ULS) must be used instead. An alternative possibility is to transform the singular matrix by a simple procedure into a positive-definite one. We propose a straightforward procedure in order to approximate an original singular matrix $R(n \times n)$ by one which is positive definite. Let $R = PQP'$ be the eigenvalue decomposition of R, where $P(n \times n)$ is the column matrix of eigenvectors and $Q = \text{diag}(q_1, \ldots, q_n)$, the matrix of eigenvalues. Let q_1 be the concerning zero eigenvalue. Then a plausible positive-definite approximation R^* to R is obtained by fixing q_1 at a small positive value p. Accordingly, $R^* = PQ^*P'$, where $Q^* = \text{diag}(p, q2, \ldots, q_n)$. Although one would like to take $p > 0$ as small as possible, efficient values of p are bounded from below due to LISREL's finite computational precision. By some trial and error it was found that $p = .02$ is close to this lower bound while still yielding an R^* which is acceptable to LISREL.

There are several advantages to using ML estimation instead of ULS. First, ML is better conditioned and therefore much faster than ULS, and second, ML is scale invariant, whereas ULS is not (Timm, 1975, pp. 557–560). Even if ML estimation is used, however, normal theory standard errors and χ^2 goodness-of-fit measures should not be used for input matrices of tetrachloric and polyserial correlations (Jöreskog and Sörbom, 1986b, p. IV6) or for matrices that are nearly singular (Boomsma *et al.*, 1989). Alternatives to a formal test of the model are to look at matrices of fitted moments, fitted residuals, and normalized residuals, to get an idea of the discrepancy between the observed and the estimated input matrices. Normalized residuals that are larger than 2 (absolute value) are indicative of specification errors in the model. Relatively large values of first-order derivatives also indicate which part of the model does not fit very well. The adjusted goodness-of-fit index (AGFI) may be used as an overall goodness-of-fit measure for each separate group in a multisample analysis (Jöreskog and Sörbom, 1986b, p. I40, V4). AGFI is independent

of sample size and relatively robust against departures from normality. It should be between zero and one, and relatively larger values indicate a better fit.

RESULTS

Means

Mean ages of twins and parents are given in Table I. There was a small, but statistically significant difference in age ($F_{4,175} = 2.87$, $p = .03$) among the five twin groups, identical twins of both sexes being somewhat younger than fraternal twins. There also is a difference between the ages of parents of MZ and those of DZ twins ($F_{4,175} = 3.76$, $p = .005$), parents of identical twins being almost 4 years younger than parents of fraternal twins. This may partly reflect the increase in DZ twinning rate

Table I. Univariate Statistics for Age, Sports Participations, and Heart Rate, for Father, Mother, Twin 1, and Twin 2 of MZ and DZ Male and Female Twin Families

	Age [mean (SD)]	Sport yes	Sport threshold	Heart rate (interbeat intervals) [mean (SD)]
MZF (28)				
Fa	47.2 (5.7)	13	.09	974 (113)
Mo	43.9 (5.4)	12	.18	891 (113)
T1	16.3 (2.3)	20	−.57	891 (124)
T2		17	−.27	899 (140)
MZM (16)				
Fa	46.9 (6.9)	7	.16	1001 (180)
Mo	44.5 (6.3)	8	.00	904 (129)
T1	17.3 (2.0)	13	−.89	927 (167)
T2		13	−.89	906 (154)
DZF (17)				
Fa	51.3 (7.4)	6	.38	957 (119)
Mo	48.8 (7.3)	5	.54	865 (130)
T1	17.7 (2.4)	13	−.72	906 (141)
T2		11	−.38	871 (130)
DZM (15)				
Fa	50.2 (5.9)	4	.62	851 (113)
Mo	47.7 (4.7)	6	.25	934 (132)
T1	17.4 (1.7)	12	−.84	931 (149)
T2		11	−.62	882 (172)
DZOS (14)				
Fa	50.6 (7.5)	4	.57	897 (131)
Mo	45.8 (6.1)	3	.79	895 (121)
Females	16.9 (1.8)	11	−.79	942 (189)
Males		11	−.79	1000 (172)

with maternal age (Bulmer, 1970). This difference does not reach statistical significance, however, for fathers ($F_{4,85} = 1.81, p = .13$) or mothers ($F_{4,85} = 2.34, p = .06$) separately. The number of "yes" responses to the sports question is shown next in Table I. There were no sex differences in sports participation for twins ($\chi_1^2 = 1.6, p = .2$) or for parents ($\chi^2 = 0$), nor were there any differences in sport participation among five groups for twins ($\chi_4^2 = 3.2, p = .52$) or for parents ($\chi_4^2 = 4.8, p = .37$). Sports involvement was less, however, for parents than for their children ($\chi_1^2 = 45.2, p = .00$). This is also reflected in the thresholds for the underlying continuous distribution, as estimated by LISREL and shown in Table I. In LISREL it is not possible to constrain these thresholds to be equal for Twin 1 and Twin 2 or for MZ and DZ groups. The estimated thresholds indicate a lower value for children than for parents, reflecting that children are more involved in sports activities than their parents.

Finally, Table I shows the mean interbeat intervals for all groups. For the children there were no significant differences among groups ($F_{4,175} = 1.5, p = .21$), although we note that both male and female opposite-sex twins have larger interbeat intervals (and thus lower heart rates) than any of the other twin groups. There also was no mean difference in IBI among parents of these five groups ($F_{4,175} = 1.1, p = .37$). Analysis of variance with sex and generation as factors showed a significant main effect of sex ($F_{1,358} = 6.7, p < .01$). Post hoc analyses revealed this difference in heart rate to be significant between fathers and mothers ($F_{1,178} = 5.7, p = .018$), but in the children's generation the difference between males and females did not reach statistical significance ($F_{1,179} = 1.8, p = .18$). The main effect of generation ($F < 1$) and the interaction of sex and generation ($F < 1$) were also not significant. Using data that are not independent may create a bias in significance testing. Therefore, four subsamples were created by randomly selecting one father, mother, son, and daughter from different families. This procedure, of course, create a loss of power and only two of the four ANOVAs now gave a main effect of sex on heart rate, while no effects of generation or of generation $\times$ sex were observed.

Correlations

Table II shows phi coefficients, tetrachoric correlations, and concordance data for sports participation and product–moment correlations for heart rate for twins, for spouses, and for parents and their offspring regardless of the zygosity of the offspring. Phi coefficients for the association of sports involvement among family members are somewhat lower than tetrachoric correlations (see Lord and Novick, 1968, p. 347), but the

Table II. MZ and DZ, Spouse–Spouse, and Parent–Offspring Correlations for Sports Participation and Heart Rate (HR)

	Sport				Sport and HR phenotypic (polyserial) correlation[a]	
	Phi coefficient	Tetrachoric	Concordance	HR		
MZF (28)	.79*	.90*	25/28	.73*	.08	.43*
MZM (16)	1.00*	.89*	16/16	.79*	.53*	.34
DZF (17)	.46	.70*	13/17	.71*	−.19	.01
DZM (15)	.08	.14	10/15	.55*	.01	.46*
DZOS (14)	−.28	−.02	8/14	.14	.69*	.66*
Fa–Mo (90)	.24*	.38*	58/90	.08	.06	.20
Fa–Son 1[b] (31)	.08	.14	14/31	.22		.24
Fa–Son 2[c] (45)	.24	.48*	22/45	.27		.51*
Fa–Daug 1[d] (59)	.21	.34*	33/59	.08		.17
Fa–Daug 2[e] (45)	.31*	.54*	27/45	.21		.27
Mo–Son 1[b] (31)	.18	.32	17/31	.40*		
Mo–Son 2[c] (45)	.16	.31*	22/45	.15		
Mo–Daug 1[d] (59)	.29*	.49*	34/59	.22		
Mo–Daug 2[e] (45)	.26	.47*	25/45	.17		

[a] Phenotypic correlations are given for Twin 1 and Twin 2, fathers, mothers, and youngest and oldest sons and daughters.
[b] All youngest sons.
[c] All oldest sons plus all OS sons.
[d] All youngest daughters plus all OS daughters.
[e] All oldest daughters.
* $p < .05$.

agreement between tetrachoric correlations and phi coefficients is quite reasonable. Although the estimates of tetrachoric correlations obtained in LISREL appear to be reliable [as also indicated by their similarity to tetrachoric correlations obtained with other programs such as BMDP4F (1985)], this does not apply to estimates for the most extreme value of $r = 1$. For MZ males the tetrachoric correlation is 1 and LISREL gives an estimate that is too low, which clearly constitutes a program error.[3]

For sports involvement, the pattern of tetrachoric correlations for female twins suggests an important contribution of shared environmental factors. In males, in contrast, common environmental influences seem to be entirely absent, as the DZ correlation is much lower than the MZ correlation and even lower than correlations between parents and offspring. Based on the observed correlations of twins only, the contribution of genetic and nongenetic factors would seem different in males and fe-

[3] This has been put right in PRELIS 1.8 (Jöreskog, personal communication).

males. The correlation of OS twins is zero, which suggests that different factors may contribute to variance in sports participation in boys and girls. There is, however, no indication of a sex difference in heritability when we look at parent–offspring correlations. There is a relatively high correlation between spouses.

In contrast, the correlation for heart rate between spouses is low. In both male and female twins there is some influence of shared environment on heart rate, as DZ correlations are higher than half the MZ correlation. Correlations of opposite-sex (OS) twins are lower than the DZ same-sex correlations, however, and the average correlation between parents and children is also lower than the DZ correlation. The association between sports and heart rate, finally, is about .3 in twins, is .2 in mothers, and is almost absent in fathers.

Model Fitting

Observed variables in twins and their parents were represented by Y variables in LISREL; latent genotypes (G) and environments (E) were represented by η variables. Causal effects of η variables on other η variables are specified in the beta matrix.

Genetic resemblance between parents and twins is accounted for by a path in B from GF and GM to $G1$ and $G2$, which is fixed at .5. $G1$ and $G2$ are correlated .5 in DZ and 1.0 in MZ twins. These correlations are specified in Ψ, the correlation matrix of the residual part of the latent η factors, i.e., that part of η that is not explained by other latent factors in the model. The variance of the residuals of $G1$ and $G2$ equals .5 (i.e., $1 - .5^2$ var $GF - .5^2$ var GM). The covariance of these residuals is zero for DZ twins, as their genetic resemblance is fully explained by their genetic resemblance to their parents. MZ twins are genetically identical, so the covariance between their genetic residuals equals the variance of these residuals. The correlation matrix of η factors can be requested on the output card, so that these specifications can be verified. The same reasoning applies to the variances of the E factors in the children, with the exception that here the path from E in the parents to E of the children is unknown. This path is estimated in B and the variance of the E residual, which is equal to $1 - 2z^2[1 + \psi(s)]$, also has to be a free parameter. Environmental resemblance of twins that is not accounted for by their environmental resemblance to their parents to estimated by $\psi(t)$ in the Ψ matrix and the correlation between EM and EF is also estimated in Ψ. A simulation study was carried out in which the following values for parameters were used: $h^2 = .3$ ($h = .5477$), $e^2 = .7$ ($e = .8366$), the correlation between total environments of spouses was .8, the residual twin

correlation was .3, and the path from parental environment to child's environment was .4. The LISREL model and the matrices generated by these parameter values are given in the Appendix (Fig. A1). Good starting values for LISREL are starting values that are consistent with the model. This is easily accomplished by having starting values of zero for most parameters, with the exception of h, e, and the residual variances in Ψ. LISREL estimates were exactly the same as true parameter values, and the input matrices were exactly replicated so that all fitted residuals were zero.

A second analysis with the same model was carried out, but now with input matrices from the Eaves *et al.* (1989) simulation study (allowing transmission paths from *EF* and *EM* to *ET* to be different). Here, also, zero residuals and an exact replication of the input matrices were obtained; indicating an exact fit to matrices that were generated according to a different model. Estimates obtained were $h = .548$, $e = .837$, $f = .147$, $m = .311$, and var res(ET) = .819. The correlation between environments of spouses was .686, which gives an observed correlation of .7 * .686 = .48. The correlation of residual environments in children was .676, and the resemblance between their environments that is caused by resemblance to their parent's environment, $f^2 + m^2 + 2fm\psi(s) = .181$, so that the estimate of shared environment in children is (.676 + .181) * .7 = .6, which is identical to the c^2 of .6 that was used to simulate the data. These results suggest that the two models are equivalent.

Several more analyses were carried out with extreme parameter values (e.g., negative transmission paths) and no differences between input matrices and fitted moments were observed.

Univariate Analyses

Sports

The full model as described above was first fitted to data of female twin families. Table IIIA shows the squared estimates of h and e, the environmental transmission path, and the correlations between environments of spouses and environments of twins. Table IIIA also shows the adjusted goodness-of-fit index for each sex/zygosity group. For female twins and their parents the largest part of the variance in sports participation is explained by environmental factors. These are shared between the parents, as is reflected in a quite high correlation between total parental environments. The total correlation between environments of the

twins was .85 (this correlation is given in the matrix of η factors) so that c^2 for female twins equals .85 * .65 = .55. The largest part of this correlation (.69) does not depend on the resemblance of offspring environment to parental environment but represents shared environmental influences unique to twins.

As there is no evidence for shared environment for male twins, a model with only additive genetic, random environmental factors and a correlation between environments of spouses was specified. This resulted in an estimate of the spouse correlation that was slightly larger than 1. This correlation was therefore fixed at 1 and this model gave a good approximation to the input matrices, as judged by the fitted residuals and AGFI.

Next, data of male and female twins were combined into one analysis. By constraining the factor loadings of observed variables on latent genetic and environmental factors to be equal across all groups, genetic and total environmental influences are forced to be the same for males and females. Based on the different heritability estimates for male and female twins, this might not seem very realistic, but the pattern of correlations between parents and offspring is the same in both sexes. Moreover, the adjusted goodness-of-fit indices for females in the first analysis are not very high and the fitted residuals indicate that the largest discrepancy between observed and estimated correlations is for parent–offspring correlations, suggesting that heritabilities are too low to account adequately for the observed parent–offspring correlations. Hence, a model where the genetic structure is the same in both sexes seems plausible. Table IIIA shows that AGFI for all groups improves when male and female data are thus combined into one analysis. To model shared environment for female twins, environmental transmission paths and the residual environmental correlation were free to be estimated, while these were zero for male twins. The estimate for environmental transmission from parents to girl twins now is lower than in the first analysis, as their resemblance is adequately explained by genetic resemblances, while the estimate of shared environment between female twins stays high. The last row in Table IIIA shows the result of adding data of OS twins to this model. As the OS twin correlation for sports participation is zero, the estimate for the correlation between environmental influences in this group becomes negative, thereby counteracting the positive genetic correlation. This suggests that, under the assumption that genetic factors are the same in both sexes and their effects of equal magnitude in males and females, environmental influences relevant to variation in sports participation are different in males and females. For female twins these environmental influences are, to a

Table III. Univariate Analyses: (A) Sports Participation (Based on Tetrachoric Correlations) and (B) Heart Rate (Based on Covariances)[a]

(A) Sports

	h^2	e^2	$\psi(s)$	z		$\psi(t)$		OS
				F	M	F	M	
1. Females	.35	.65	.81	.21	—	.69	—	—
2. Males	.77	.23	1[b]	—	—	—	—	—
3. Fe + Ma	.64	.36	1[b]	.07	—	.84	—	—
4. Fe + Ma + OS	.64	.36	1[b]	.03	—	.80	—	−.42

AGFI

	MZF	DZF	MZM	DZM	OS
1	.84	.82	—	—	—
2	—	—	.93	.94	—
3	.92	.93	.96	.98	—
4	.96	.95	.96	.98	.97

(B) Heart rate

	LISREL estimate (SE in parentheses)								
	h	e	$\psi(t)$	h^2	e^2	V_p	x^2	df	p
1. Females	68	108	.62	.28	.72	16381	20.9	17	.23
	(20)	(13)	(.11)						
2. Males	110	101	.47	.55	.45	22238	13.1	17	.73
	(20)	(17)	(.21)						
3. Fe + Ma	88	105	.56	.42	.58	18745	38.0	37	.42
	(14)	(10)	(.11)						
4. Fe + Ma + OS	96	105	.46	.45	.55	20072	53.6	47	.24
	(12)	(09)	(.12)						

[a] z is the environmental transmission from parents to F(emale) and M(ale) offspring, $\psi(s)$ is the correlation between total environments of spouses, $\psi(t)$ is the correlation between residual environments of twins and may be different for F(emale), M(ale), and OS twin pairs. V_p is the total phenotypic variance.

[b] Parameter fixed at this value.

large extent, shared between them, and for OS twins some sort of negative sibling interaction might be present.

Heart Rate

Univariate analyses of heart rate were carried out on covariance matrices. For these analyses it is therefore permissible to use standard errors and χ^2 tests. A model with h, e, and $\psi(t)$ to account for shared environment of twins was first fitted to male and female data separately and next to combined male and female data and to data from all five groups. The results are shown in Table IIIB. Estimates of heritabilities in males and females are not significantly different as judged by the fit of the third model, where male and female data were combined into one analysis. Although there is an increase in χ^2 when data from both sexes are analyzed jointly, and also when the DZOS group is added to the analysis; this increase is accompanied by a relatively large increase in df so that a nonsignificant chi-square is obtained. The same analyses as summarized in Table IIIB were also carried out on correlations instead of covariances, as these are required for the bivariate analyses of sports participation and heart rate, and almost identical results were obtained.

Bivariate Analysis

The model used for the univariate analyses can be extended to the bivariate case in several ways. One approach is the factor model of Martin and Eaves (1977), where correlations between variables arise because the variables have loadings on the same genetic and/or environmental factors. Using this approach, correlations between spouses can be modeled by a correlation between general factors of father and mother. Assuming that a correlation between two observed variables X and Y is caused by their loadings on the same latent environmental factor, i.e., $X = E + \epsilon_x$ and $Y = E + \epsilon_y$, where E represents an environmental factor that is common to both X and Y and ϵ is a unique factor, then a correlation $\psi(s)$ between environments of father (EF) and mother (EM) creates the following phenotypic correlations between spouses: for X (and Y), $\psi(s)e_x e_x$. The correlation between X in father and Y in mother (or Y in mother and X in father) becomes $\psi(s)e_x e_y$ (where e_x and e_y are factor loadings on E). If this last correlation is not symmetric (i.e., X in father with Y in mother is not equal to X in mother with Y in father), an alternative representation using the beta matrix is possible, using the path from EF to EM that differs from the path EM to EF. This way spouse correlations are restricted to arise from the communal part of the model. A similar restriction could be applied to environmental transmission paths or correlations between

environmental influences in twins. Table IV illustrates some of these ideas. Two bivariate models are considered: in the first model sports participation and heart rate load on a general genetic factor, and in the second model both variables load on the same environmental factor. In addition, the first model specifies unique environmental factors for sports and heart rate and a unique genetic factor for sports only. Based on the results of univariate analyses, there is a correlation between environmental influences relevant to variation in sports participation for spouses, female twins, and OS twins. For heart rate, no spouse correlation and an equal correlation between environments were specified for all twins. The second model explains the association between sports participation and heart rate by their loadings on a general environmental factor that is correlated in spouses and female and OS twins. Inspection of Table IV shows that the two models yield almost identical AGFIs. It turns out that the general genetic factor in the first model is in fact a heart-rate factor (as indicated by a low loading of sports and a relatively high loading of heart rate). In the second model, the general environmental factor is in fact a sports factor. Hence, we seem to have four factors that are relatively independent. This probably is a fair picture, as the correlations of heart rate and sports participation are not very high.

DISCUSSION

Our data indicate that variation in sports participation is influenced to a large extent by genetic factors. In view of the fact that variation in sports performance is determined almost exclusively by genetic factors (e.g., Malina and Bouchard, 1986), it does not seem unlikely that the choice to participate in sports activities would also have a genetic component. For female twins a large part of the environmental variance is shared between them but is not shared with their parents. For boys there is no evidence for common environmental factors. The model employed assumes that assortment for sports participation is entirely environmental, and a high correlation between latent environmental factors of spouses was found. The most important limitation of analyzing any parent-offspring model in LISREL, however, is that assortment based on parental phenotypes cannot be considered, since it involves the use of nonlinear constraints (Fulker, 1982; Boomsma and Molenaar, 1987).

Results for resting heart rate are in good agreement with previous studies that used twins only. Roughly half of the variance is explained by genetic factors, and for the twins the other half is equally divided between common and random environmental factors. These estimates do not change much when parents of twins are included in the research de-

Table IV. Bivariate Analyses of Sports Participation and Heart Rate (HR) (Based on Tetrachoric, Product-Moment, and Polyserial Correlations)

	Common *G* model							
	Factor loading					Correlation of environments		
						$\psi(t)$		
	G(common)	*G*(sports)	*E*(sports)	*E*(*HR*)	$\psi(s)$	F	M	OS
Sports	.267	.748	.602	—	1[a]	.854	—	−.912
HR	.661	—	—	.751	—	.513	.513	.513
	Common *E* model							
	Factor loading					Correlation of environments		
						$\psi(t)$		
	G(sports)	*G*(HR)	*E*(common)	*E*(HR)	$\psi(s)$	F	M	OS
Sports	.790	—	.612	—	.807	.878	—	−1.0[a]
HR	—	.657	.280	.701	—	.552	.552	.552

	AGFI				
	MZF	DZF	MZM	DZM	OS
Common *G*	.96	.95	.96	.93	.89
Common *E*	.96	.94	.95	.93	.89

[a] Parameter fixed at this value.

sign. Resemblances of parents and offspring can be accounted for solely on the basis of their genetic resemblance to each other and there is no evidence for assortative mating.

Both for sports participation and heart rate, no significant sex difference in heritability was found. This is due partly to the small sample size and we may conclude only that in this sample such effects could not be detected.

APPENDIX

```
DA NG=2 NI=4 NO=100 MA=KM
LA
*
'Twin1' 'Twin2' 'FA' 'MO'
KM SY
*
1.000
 .913    1.000
 .654     .654    1.000
 .654     .654     .560    1.000
MO NY=4 NE=8 PS=SY,FI LY=FU,FI BE=FU,FI TE=ZE
LE
*
'GT1' 'GT2' 'GMO' 'GFA' 'ET1' 'ET2' 'EMO' 'EFA'
PA LY
*
1 0 0 0  1 0 0 0
0 1 0 0  0 1 0 0
0 0 1 0  0 0 1 0
0 0 0 1  0 0 0 1
EQ LY(1,1) LY(2,2) LY(3,3) LY(4,4)
EQ LY(1,5) LY(2,6) LY(3,7) LY(4,8)
FR BE(5,7) BE(5,8) BE(6,7) BE(6,8)
EQ BE(5,7) BE(5,8) BE(6,7) BE(6,8)
ST .5 BE(1,3) BE(1,4) BE(2,3) BE(2,4)
ST .5 PS(1,1) PS(2,2) PS(2,1)
ST 1  PS(3,3) PS(4,4) PS(7,7) PS(8,8)
FR PS(5,6) PS(7,8) PS(5,5) PS(6,6)
EQ PS(5,5) PS(6,6)
ST 0.7 LY(1,1) LY(2,2) LY(3,3) LY(4,4)
ST 0.7 LY(1,5) LY(2,6) LY(3,7) LY(4,8)
ST 1.0 PS(5,5) PS(6,6)
OU NS
DZ Twins and Parents
DA NO=100 MA=KM
LA
*
'Twin1' 'Twin2' 'FA' 'MO'
KM SY
*
1.000
 .763    1.000
 .654     .654    1.000
 .654     .654     .560    1.000
MO NY=4 NE=8 PS=SY,FI LY=IN BE=IN TE=ZE
LE
*
'GT1' 'GT2' 'GMO' 'GFA' 'ET1' 'ET2' 'EMO' 'EFA'
ST .5 PS(1,1) PS(2,2)
ST 1  PS(3,3) PS(4,4) PS(7,7) PS(8,8)
FR PS(5,6) PS(7,8) PS(5,5) PS(6,6)
EQ PS(1,5,6) PS(5,6)
EQ PS(1,7,8) PS(7,8)
EQ PS(1,5,5) PS(5,5) PS(6,6)
OU SE RS MR PC NS
```

Fig. A1. MZ twins and parents: simulation.

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